the abdominal cavity of rabbits, and in all instances the pulmonary vessels were found to contain numerous fatty emboli. In performing these experiments, it was observed that rapid injection of the oil is more fatal than a slow injection. When injected into a vein close to the heart, an amount of oil which, introduced under high pressure, would cause immediate death, if injected slowly produces scarcely recognizable effects. The fatty emboli are bland, and are not, as Wagner supposed, causes of pyæmia or secondary abscesses. The fat does not pass through the walls of the capillaries, but the plug becomes loosened and carried on in the general circulation, as formerly described.

With regard to the quantity of fat required to kill an animal, he comes to the conclusion that varying amounts will be necessary, according to the *vis à tergo* and the distance of the point of entrance of the fat into the circulation from the heart. If the *vis à tergo* be but little or if the distance be great, so that the increased blood-pressure produced by the entrance of the fat is equalized before it reaches the heart, a healthy animal requires a very large quantity of fat to kill it. The shorter the road, and the greater the *vis à tergo*, the less is the quantity of fat required; but in no case does Scriba think that less than three times the amount of fat present in the femur of the animal would be able to cause death. On examining the animals which died of fatty embolism, it was found that the vessels of the brain were very markedly blocked with fat; while, when these emboli were absent, or only present in small numbers, death did not occur, even although the lungs were full of them. In no case did he find death occur from acute œdema of the lungs. He therefore concludes that, in pure fatty embolism, death occurs only in consequence of extensive embolism of the vessels of the brain and spinal cord, and is due to the arterial anæmia and consequent disturbances of nutrition produced by the emboli.

He gives the following as the characteristic symptoms of fatty embolism: presence of fat in the urine at intervals; transient attacks of dyspnœa; lowering of temperature; temporary slight hæmoptysis, without fever, and generally without dulness or *râles;* irregular action of the heart; collapse, with marked pallor of the skin and mucous membranes; at first, shallow respiration, at times interrupted by deep sighing inspiration, later Cheyne-Stokes's respiration; spasms of various kinds or paralyses, generally bilateral; diminution of reflex irritability.

Such are some of the chief facts made out by Scriba with regard to the phenomena of fatty embolism. The subject is certainly one of the greatest importance, and calls for further investigation. It is, indeed, strange that, in spite of the interest which it has excited abroad, it should have attracted but little attention in this country.—*British Med. Journal*, May 22, 1880.

Starvation Fever.

At a late meeting of the College of Physicians of Philadelphia Dr. J. M. Da Costa reported three cases, occurring in the same family, of what he proposes to call "Starvation Fever."

The first case was a little girl, three years of age, pale, weakly, and irritable, who was seized, Jan. 1, with vomiting. She was very feeble, unable to sit up in bed, and complaining of giddiness. The next day the same symptoms persisted, and the pulse was small and very rapid. Subsequently there was marked fever, and the vomiting lessened; under quinia, beef-tea, brandy, and milk, the gastric symptoms disappeared. The fever was unattended with sore throat, eruption, or diarrhœa, was not of long duration, and by the ninth day from the beginning of the attack, the little patient, in good spirits, with appetite fully returned, was sitting up in bed playing, seemingly quite convalescent. On the 10th, vomiting returned; henceforth nothing was retained on the stomach; the child complained of not being able to see, became weaker and weaker, and expired exhausted on the 13th. The *autopsy* showed yellowish serous effusion in both pleural sacs. There was no evidence of lymphy exudation on the pleura; the amount of fluid was sufficient to have compressed the lower lobes of both lungs; the upper lobes were healthy. The heart was normal. The stomach was empty, its mucous membrane dryish and very pale; the intestines exhibited no swelling of any of the glands. The spleen was slightly enlarged; the liver had a nutmeg appearance; the kidneys were normal. The dura mater was somewhat congested, but free from adhesions; the pia mater was opaque; the cortical portions of the brain were œdematous; the convolutions were deep. A section of the upper part of the brain showed punctiform injection; the lateral ventricles were dry; but there was some effusion in the fourth ventricle.

The second case, a younger brother, four months of age, also a pallid, ill-nourished child, was seized with convulsions Jan. 10th. There was no retraction of the head; the urine was not albuminous. He had had a convulsive seizure two months previously, leaving apparently no trace. The convulsions of the 10th lasted until nearly evening, and some hours afterwards death took place. In the afternoon a few dark spots, like very small petechiæ, were noticed on the neck, and shortly before death similar spots were detected in the hypogastric region. The *autopsy* gave results similar to those in the first case. In all the thoracic and abdominal organs, evidences of profound anæmia; some serous effusion in the pleural sacs; stomach pale, containing a small plug of casein; absence of signs of glandular enlargement in the pale intestines, and a rather fluid blood, were the points specially noticed. Permission to examine the head was not obtained.

The third case was a little girl a year and a half old. She, too, appeared ill nourished, and was on Jan. 8th attacked with fever, with which was soon associated an acute bronchitis, and subsequently a circumscribed broncho-pneumonia. There was no sore throat, eruption, or albuminous urine. The child was well sustained, and under quinia and stimulating expectorants she slowly recovered in two weeks, having been very ill.

Dr. Da Costa was at first at a loss as to the nature of these strange cases. That they were not cases of scarlet fever, or diphtheria, or typhoid fever, was self-evident, though it was equally evident that in some general cause producing profound alteration of nutrition, or in some blood poison, was to be sought the explanation. The convulsions, the eruption, the speedy termination in the second case, suggested cerebro-spinal fever; but spinal symptoms were wanting, and the phenomena in the other cases were not at all those of this disease. The question of poisoning from bad drainage was carefully considered; the drains were everywhere in good order, and there was no water-closet or stationary wash-stand near the rooms in which the children played and slept. The supply of water in the house was good, and none of the adults suffered in the least from its free use. Attention was then directed to the food, and here a clue was obtained, which led to an unexpected discovery. The children lived almost entirely on cow's milk; the two youngest, exclusively; the eldest, almost exclusively; and, though taking large quantities, they did not thrive. Chemical analysis showed that the milk was skimmed, and diluted with one and a half parts of water.

The nature of the cases then became clear. In the anomalous fever, the local congestions, the vomiting, the stains on the skin, the convulsions, the ready collapse; in the collection of fluids within the serous sacs, the thinness and pallor of the mucous membranes, were the phenomena of inanition or starvation, as they have been experimentally demonstrated. To call these cases "Starvation

Fever,'' would, Dr. Da Costa thinks, be fully justified. Yet the question remains, whether some cause, in itself of little importance, did not act as an exciting cause to bring about the crash; some cause like a passing influenza, which, throwing the least weight on functions and organs incapable of resistance, broke them down at once. The fact of all the cases happening about the same time, and the catarrhal symptoms in the third case, favour this view.

Salicin in Acute Rheumatism.

At a late meeting of the Clinical Society of London (*Lancet*, May 29, 1880), Dr. Greenhow, President of the Society, reported ten cases of rheumatic fever treated with salicin. He stated that for some years past he has been in the habit of testing carefully and methodically in a series of cases the several remedies from time to time proposed for the treatment of rheumatic fever. During the last three years he had treated in the Middlesex Hospital ten cases with salicin, and fifty with salicylate of soda. All the patients were confined to bed until the fever had entirely subsided. The diet consisted of milk, beef-tea, and sometimes eggs; stimulants were sparingly given; the affected joints were wrapped in cotton-wool, and, when pericarditis or endocarditis was present, belladonna and iodide of potassium ointment was applied over the præcordia. In most cases simple effervescing medicine was prescribed on the day of admission, and aperients and occasionally sedatives were given, but the latter as seldom as possible. In all the cases treated with salicin, and in many of those treated with salicylate of soda, the urine was examined the day after beginning the medicine, and found invariably to yield the characteristic reaction with perchloride of iron. In order to exclude cases of mild type which, especially in young subjects, rapidly improve under any kind of treatment, the salicin or salicylate was not prescribed until the patient had been from twenty-four to thirty-six hours in the wards.

Case 1. First attack. Incipient pericarditis. Salicin in full doses on day after admission. Temperature began to fall on third day, became normal on sixth. Pains abated on second day, and ceased on fifth. The salicin taken every two hours for six days, then for two days every four hours, and for nine days every six hours. A mild relapse occurred whilst taking the drug. Epistaxis occurred in this case, and the heart's action became feeble, requiring stimulants. Discharged on sixty-sixth day. Case 2. Sixth attack. Admitted on fourteenth day. Mitral disease and pericarditis. Pain diminished on second day of treatment, ceased on sixth day. Temperature fell on third day, and became normal on fifth. Salicin given every two hours for six days, then every four hours, but, a relapse occurring, the frequency was again increased to two hours. Vomiting and headache became so severe that, at the end of ten days more, the drug was discontinued. Pleurisy and a second attack of pericarditis occurred. Discharged on 107th day. Case 3. First attack. Ailing several weeks. Pericarditis. Pains rapidly subsided and temperature became normal on the fourth day of taking the drug, but cardiac failure was very marked, and the salicin was discontinued on the eighth day. Discharged with an apex systolic murmur on twenty-fifth day. Case 4. Third attack. Old mitral disease, and a recent basic murmur. Pericarditis supervened two days after salicin commenced. The temperature soon fell to normal, but pains and vomiting did not subside with it. A mild relapse occurred after the salicin was discontinued; iodide of potassium was given, and patient discharged on fifty-eighth day. Case 5. Third attack; relapse; pericarditis. Temperature normal, and pains greatly subsided on fourth day. Epistaxis supervened, and pulse became small and feeble on the third day. Discharged on twentieth day. Case 6. First attack